	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
1	BRS	L1	82	clostridial adj neurotoxin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/30 12:32			0
2	BRS	L2	429	botulinum adj toxin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/30 12:34			0
3	BRS	L3	137	transmission adj compound	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/30 12:35			0
4	BRS	L4	18077	glutamate	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/30 12:35			0
5	BRS	L5	5284	(substance adj P) or (calcitonin adj gene adj related adj peptide) or (neuropeptide adj y)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/30 12:37			0
6	BRS	L6	1268	target$3 adj moiety	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/30 12:38			0
7	BRS	L7	12	(3 or 4 or 5) same 6	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/30 12:38			0
8	BRS	L8	79	(1 or 2) same (recombinant or express$3)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/30 12:39			0
9	BRS	L9	1	7 same 8	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/30 12:40			0

=> d his

(FILE 'HOME' ENTERED AT 12:44:17 ON 30 OCT 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
12:44:46 ON 30 OCT 2002
L1 871 S CLOSTRIDIAL NEUROTOXIN
L2 17567 S BOTULINUM TOXIN
L3 569 S (L1 OR L2) (P) (RECOMBINANT OR EXPRESS?)
L4 626 S TARGET? MOIETY
L5 12 S TRANSMISSION COMPOUND
L6 103866 S (SUBSTANCE P) OR TACHYKININ
L7 73957 S (CALCITONIN GENE RELATED PEPTIDE) OR (NEUROPEPTIDE Y)
L8 3 S L4 (P) (L5 OR L6 OR L7)
L9 0 S L3 (P) L8
L10 3 DUPLICATE REMOVE L8 (0 DUPLICATES REMOVED)
L11 20 S L3 (P) (L5 OR L6 OR L7)
L12 5 DUPLICATE REMOVE L11 (15 DUPLICATES REMOVED)
L13 5 S L12 NOT L10

=> log y

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FILE 'HOME' ENTERED AT 12:44:17 ON 30 OCT 2002

=> file medline caplus biosis embase scisearch agricola
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

FILE 'MEDLINE' ENTERED AT 12:44:46 ON 30 OCT 2002

FILE 'CAPLUS' ENTERED AT 12:44:46 ON 30 OCT 2002
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FILE 'AGRICOLA' ENTERED AT 12:44:46 ON 30 OCT 2002

=> s clostridial neurotoxin
L1          871 CLOSTRIDIAL NEUROTOXIN

=> s botulinum toxin
L2        17567 BOTULINUM TOXIN

=> s (l1 or l2) (p) (recombinant or express?)
L3          569 (L1 OR L2) (P) (RECOMBINANT OR EXPRESS?)

=> s target? moiety
L4          626 TARGET? MOIETY

=> s transmission compound
L5           12 TRANSMISSION COMPOUND

=> s (substance P) or tachykinin
L6       103866 (SUBSTANCE P) OR TACHYKININ

=> s (calcitonin gene related peptide) or (neuropeptide y)
L7        73957 (CALCITONIN GENE RELATED PEPTIDE) OR (NEUROPEPTIDE Y)

=> s l4 (p) (l5 or l6 or l7)
L8            3 L4 (P) (L5 OR L6 OR L7)

=> s l3 (p) l8
L9            0 L3 (P) L8

=> duplicate remove l8
PROCESSING COMPLETED FOR L8
L10           3 DUPLICATE REMOVE L8 (0 DUPLICATES REMOVED)

=> d l10 1-3 ibib abs

L10  ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:         2002:89857  CAPLUS
DOCUMENT NUMBER:          136:145260
TITLE:                    Clostridial toxin derivatives and methods for treating
                          pain
INVENTOR(S):              Donovan, Stephen
PATENT ASSIGNEE(S):       Allergan Sales, Inc., USA
SOURCE:                   PCT Int. Appl., 67 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   3
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PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002007759	A2	20020131	WO 2001-US21984	20010712

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-625098 A 20000725

AB Methods for treating a bone tumor, in particular pain assocd. with bone tumor, by administration to a patient of a therapeutically effective amt. of an agent are disclosed. The agent may include a clostridial neurotoxin component attached to a ***targeting*** ***moiety*** , wherein the ***targeting*** ***moiety*** is selected from the group consisting of ***transmission*** ***compds*** . which can be released from neurons upon the transmission of pain signals by the neurons, and compds. substantially similar to the ***transmission*** ***compds*** .

L10 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:241331 CAPLUS
DOCUMENT NUMBER: 136:273210
TITLE: Clostridial toxin derivatives and methods for treating pain
INVENTOR(S): Donovan, Stephen
PATENT ASSIGNEE(S): Allergan Sales, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 20 pp., Cont.-in-part of U.S. Ser. No. 625,098.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002037833	A1	20020328	US 2001-922093	20010803

PRIORITY APPLN. INFO.: US 2000-489667 A2 20000119
US 2000-625098 A2 20000725

AB Agents for treating pain, methods for producing the agents and methods for treating pain by administration to a patient of a therapeutically effective amt. of the agent are disclosed. The agent can include a clostridial neurotoxin, or a component or fragment or deriv. thereof, attached to a ***targeting*** ***moiety*** , wherein the ***targeting*** ***moiety*** is selected from a group consisting of ***transmission*** ***compds*** . which can be released from neurons upon the transmission of pain signals by the neurons, and compds. substantially similar to the ***transmission*** ***compds*** . The agent comprises a botulinum toxin component covalently coupled to ***substance*** ***P*** .

L10 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:545729 CAPLUS
DOCUMENT NUMBER: 135:132453
TITLE: Clostridial neurotoxin derivatives attached to targeting moieties, and methods using them for treating pain
INVENTOR(S): Donovan, Stephen
PATENT ASSIGNEE(S): Allergan Sales, Inc., USA
SOURCE: PCT Int. Appl., 76 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

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     WO 2001053336     A1   2001[redacted]6       WO 2001-US1529   200101[redacted]
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
             HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
             LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
             SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
             YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
             DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
             BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     US 2002068699     A1   20020606        US 2001-938112   20010823
PRIORITY APPLN. INFO.:                    US 2000-489667   A  20000119
AB   The invention provides agents for treating pain, methods for producing the
     agents, and methods for treating pain by administration to a patient of a
     therapeutically effective amt. of the agent.  The agent can include a
     clostridial neurotoxin, or a component of fragment or deriv. thereof,
     attached to a   ***targeting***     ***moiety***  , wherein the
       ***targeting***     ***moiety***   is selected from   ***transmission***
       ***compds***  . which can be released from neurons upon the transmission
     of pain signals by the neurons, and compds. substantially similar to the
       ***transmission***     ***compds***  .
REFERENCE COUNT:          9     THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS
                                RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT


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     (FILE 'HOME' ENTERED AT 12:44:17 ON 30 OCT 2002)

     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     12:44:46 ON 30 OCT 2002
L1           871 S CLOSTRIDIAL NEUROTOXIN
L2         17567 S BOTULINUM TOXIN
L3           569 S (L1 OR L2) (P) (RECOMBINANT OR EXPRESS?)
L4           626 S TARGET? MOIETY
L5            12 S TRANSMISSION COMPOUND
L6        103866 S (SUBSTANCE P) OR TACHYKININ
L7         73957 S (CALCITONIN GENE RELATED PEPTIDE) OR (NEUROPEPTIDE Y)
L8             3 S L4 (P) (L5 OR L6 OR L7)
L9             0 S L3 (P) L8
L10            3 DUPLICATE REMOVE L8 (0 DUPLICATES REMOVED)

=> s l3 (p) (l5 or l6 or l7)
L11           20 L3 (P) (L5 OR L6 OR L7)

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KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
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L12            5 DUPLICATE REMOVE L11 (15 DUPLICATES REMOVED)

=> s l12 not l10
L13            5 L12 NOT L10

=> d l13 1-5 ibib abs

L13  ANSWER 1 OF 5     MEDLINE
ACCESSION NUMBER:    2001325272     MEDLINE
DOCUMENT NUMBER:     21218317   PubMed ID: 11320861
TITLE:               [Botulinum toxin A for the treatment of headache disorders
                     and pericranial pain syndromes].
                     Botulinum-Toxin A in der Therapie von
                     Kopfschmerzerkrankungen und perikranialen Schmerzsyndromen.
AUTHOR:              Gobel H; Heinze A; Heinze-Kuhn K; Austermann K
CORPORATE SOURCE:    Neurologisch-verhaltensmedizinische Schmerzklinik Kiel in
                     Kooperation mit der Universitat Kiel, Heikendorfer Weg
                     9-27, 24149 Kiel.. kiel@Schmerzklinik.de
SOURCE:              NERVENARZT, (2001 Apr) 72 (4) 261-74.  Ref: 104
                     Journal code: 0400773. ISSN: 0028-2804.
PUB. COUNTRY:        Germany: Germany, Federal Republic of
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
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General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010611
Last Updated on STN: 20010611
Entered Medline: 20010607

AB For 20 years ***botulinum*** ***toxin*** A has been used for the treatment of a variety of disorders characterised by pathologically increased muscle contraction. Recently, treatment of tension headache, migraine, cluster headache, and myofascial pain syndromes of neck, shoulder girdle, and back with ***botulinum*** ***toxin*** A has become a rapidly expanding new field of research. Several modes of action are discussed for these indications. The blockade of cholinergic innervation reduces muscular hyperactivity for 3 to 6 months. Degenerative changes in the musculoskeletal system of the head and neck are prevented. Nociceptive afferences and blood vessels of the pericranial muscles are decompressed and muscular trigger points and tender points are resolved. The normalisation of muscle spindle activity leads to a normalisation of muscle tone and central control mechanisms of muscle activity. Oromandibular dysfunction is eliminated and muscular stress removed. However, the effect of ***botulinum*** ***toxin*** A cannot be explained by muscular actions only. Its retrograde uptake into the central nervous system modulates the ***expression*** of ***substance*** ***P*** and enkephalins in the spinal cord and nucleus raphe. Recent findings suggest an inhibition of sterile inflammation which may lead to a blockade of the neurogenic inflammation believed to be the pathophysiological substrate of primary headache disorders. The efficacy of ***botulinum*** ***toxin*** A in the treatment of pain disorders is being investigated in several studies at the moment. The results and experiences obtained so far present new alternatives in the treatment of chronic pain disorders. The practical use of ***botulinum*** ***toxin*** A is demonstrated.

L13 ANSWER 2 OF 5 MEDLINE
ACCESSION NUMBER: 2000148594 MEDLINE
DOCUMENT NUMBER: 20148594 PubMed ID: 10683301
TITLE: Enkephalin and aFGF are differentially regulated in rat spinal motoneurons after chemodenervation with botulinum toxin.
AUTHOR: Humm A M; Pabst C; Lauterburg T; Burgunder J M
CORPORATE SOURCE: Laboratory of Neuromorphology, University of Berne, Berne, CH3010, Switzerland.
SOURCE: EXPERIMENTAL NEUROLOGY, (2000 Jan) 161 (1) 361-72.
Journal code: 0370712. ISSN: 0014-4886.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000330
Last Updated on STN: 20020914
Entered Medline: 20000323

AB ***Botulinum*** ***toxin*** is used to induce transient graded paresis by chemodenervation in the treatment of focal hyperkinetic movement disorders. While the molecular events occurring in motoneurons after mechanical nerve lesioning leading to muscle paresis are well known, they have been investigated to a lesser extent after chemodenervation. We therefore examined the ***expression*** of enkephalin (ENK), acidic fibroblast growth factor (aFGF), neurotensin (NT), galanin (GAL), ***substance*** ***P*** (SP), vasoactive intestinal polypeptide (VIP), and ***neuropeptide*** ***Y*** (NPY) in rat spinal motoneurons after chemodenervation of the gastrocnemius. In order to precisely localize the motoneurons targeting the injection site, retrograde tracing was performed in additional rats by using Fluorogold injections. ENK ***expression*** was upregulated in the region corresponding to the Fluorogold positive motoneurons, but also on the contralateral side and in more distant parts of the spinal cord. The highest upregulation occurred 7 to 14 days after injections and decreased over a period of three months. At 8 days, aFGF was slightly downregulated in all regions studied, single motoneurons showed NT ***expression*** ,

while · ***expression*** of GAL, SP, VIP, and NPY could be detected neither in controls nor in [redacted]in-treated animals. These alter[redacted]ions in gene ***expression*** were strikingly different from those described after axotomy. Our present findings give additional demonstration of the considerable plasticity of the adult spinal cord after ***botulinum*** ***toxin*** treatment.

L13 ANSWER 3 OF 5 MEDLINE
ACCESSION NUMBER: 97291189 MEDLINE
DOCUMENT NUMBER: 97291189 PubMed ID: 9145803
TITLE: Expression of neurotransmitter genes in rat spinal motoneurons after chemodenervation with botulinum toxin.
AUTHOR: Jung H H; Lauterburg T; Burgunder J M
CORPORATE SOURCE: Neuromorphological Laboratory of the Department of Neurology, University of Berne, Switzerland.
SOURCE: NEUROSCIENCE, (1997 May) 78 (2) 469-79.
Journal code: 7605074. ISSN: 0306-4522.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 19970813
Last Updated on STN: 19980206
Entered Medline: 19970801

AB ***Botulinum*** ***toxin*** is widely used for the treatment of focal movement disorders, where chemodenervation is used to decrease hyperactivity in selected muscles. Beside a focal paresis, widespread effects on neuromuscular synaptic function have been demonstrated. However, reactions of motoneurons after neuromuscular chemodenervation without gross morphological lesions are largely unknown. Peripheral axotomy, in contrast, leads to profound changes in the ***expression*** of several genes, including those encoding neurotransmitters, in motoneurons. We therefore examined the ***expression*** of neurotransmitter genes in rat motoneurons six days after intramuscular ***botulinum*** ***toxin*** application in the right gastrocnemius muscle. Similar doses of ***botulinum*** ***toxin*** as used in human where injected. A focal bilateral increase in ***expression*** of the choline acetyltransferase gene and a widespread bilateral increase of the beta- ***calcitonin*** - ***gene*** - ***related*** ***peptide*** and the enkephalin genes was measured in motoneurons after ***botulinum*** ***toxin*** injection. Cholecystokinin had a lower ***expression*** after ***botulinum*** ***toxin*** injections. Growth-associated protein 43, nitric oxide synthase, somatostatin and proopiomelanocortin messenger RNA were not found in motoneurons of both groups. Our results demonstrate that changes in the ***expression*** of neurotransmitter genes in motoneurons also occur after chemodenervation but with different patterns to those found after mechanical nerve lesioning. These changes reflect focal and widespread modulative events. The knowledge of these events should lead to a better understanding of the focal paralysis and of the more widespread effects found in human after intramuscular injection of ***botulinum*** ***toxin*** .

L13 ANSWER 4 OF 5 MEDLINE
ACCESSION NUMBER: 97078373 MEDLINE
DOCUMENT NUMBER: 97078373 PubMed ID: 8919297
TITLE: Effect of muscle denervation on the expression of substance P in the ventral raphe-spinal pathway of the rat.
AUTHOR: Van den Bergh P; De Beukelaer M; Deconinck N
CORPORATE SOURCE: Laboratoire de Biologie Neuromusculaire, Service de Neurologie, Cliniques Universitaires St-Luc, Universite de Louvain, Brussels, Belgium.
SOURCE: BRAIN RESEARCH, (1996 Jan 29) 707 (2) 206-12.
Journal code: 0045503. ISSN: 0006-8993.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY DATE: Entered STN: 19970407
Last Updated on STN: 20000407

Entered Medline: 19970327

AB The medullary raphe nuclei, wherein serotonin (5-HT) coexists with ***substance*** ***P*** (SP) and thyrotropin-releasing hormone (TRH), innervate lower motor neurons in the spinal cord ventral horn by means of the ventral raphe-spinal pathway. Destruction of the ventral raphe-spinal pathway is associated with deficient recovery of denervated muscle, indicating that it may exert a trophic effect upon lower motor neurons. To determine whether SP could be a trophic factor for lower motor neurons within the ventral raphe-spinal pathway, the effect of muscle denervation with ***botulinum*** ***toxin*** type A on SP-encoding beta-preprotachykinin mRNA in the rat medullary raphe was examined by in situ hybridization histochemistry. Silver grain density over hybridized medullary raphe neurons was increased by up to 11%, although the number of hybridized neurons did not change in denervated as compared to control rats. Increased SP gene ***expression*** in the medullary raphe in response to motor unit lesioning suggests that raphe-spinal SP may be trophic to lower motor neurons.

L13 ANSWER 5 OF 5 MEDLINE
ACCESSION NUMBER: 95123477 MEDLINE
DOCUMENT NUMBER: 95123477 PubMed ID: 7823160
TITLE: Calcitonin gene-related peptide: possible role in formation and maintenance of neuromuscular junctions.
AUTHOR: Sala C; Andreose J S; Fumagalli G; Lomo T
CORPORATE SOURCE: CNR Center of Cytopharmacology, University of Milano, Italy.
SOURCE: JOURNAL OF NEUROSCIENCE, (1995 Jan) 15 (1 Pt 2) 520-8. Journal code: 8102140. ISSN: 0270-6474.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950223
Last Updated on STN: 19970203
Entered Medline: 19950216

AB The ***expression*** and content of ***calcitonin*** ***gene*** - ***related*** ***peptide*** (CGRP) and secretogranin II (SgII) in adult rat motor neurons were examined by in situ hybridization, Northern blot analysis, and immunocytochemistry. Normal motor nerve terminals did not contain detectable CGRP or SgII. Ten to 15 days after a peripheral nerve crush about 80% of the motor nerve terminals reinnervating the soleus (SOL) muscle contained detectable CGRP but no SgII. Thereafter, the percentage of CGRP-positive terminals declined towards zero. In the spinal cord, CGRP ***expression*** was higher than normal 1 d after a sciatic nerve crush and increased during the next few days. No increase in SgII ***expression*** was observed. Nerve blocks by tetrodotoxin (TTX) and ***botulinum*** ***toxin*** (BoTX) increased CGRP content and ***expression*** in motor neurons but had no effect on SgII. After 10 d of BoTX treatment and 33 d of TTX treatment (the longest time points studied), more than 90% of the motor nerve terminals stained for CGRP. The density of large dense core vesicles (LDCVs) was also higher than normal in such terminals. Some increase in CGRP content and ***expression*** occurred in the nontreated side. In a group of rats, the peroneal nerve was stimulated electrically with brief, intermittent pulse trains at 100 Hz. The stimulation was applied below a TTX block that had started 7 or 19 d earlier. One minute of such stimulation was sufficient to remove CGRP from most of the terminals. (ABSTRACT TRUNCATED AT 250 WORDS)

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(FILE 'HOME' ENTERED AT 12:44:17 ON 30 OCT 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 12:44:46 ON 30 OCT 2002

L1 871 S CLOSTRIDIAL NEUROTOXIN
L2 17567 S BOTULINUM TOXIN
L3 569 S (L1 OR L2) (P) (RECOMBINANT OR EXPRESS?)
L4 626 S TARGET? MOIETY
L5 12 S TRANSMISSION COMPOUND
L6 103866 S (SUBSTANCE P) OR TACHYKININ

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L7        73957 S (CALCITONIN GENE RELATED PEPTIDE) OR (NEUROPEPTIDE Y)
L8            3 S L4 (P) (L5 OR [redacted]OR L7)
L9            0 S L3 (P) L8
L10           3 DUPLICATE REMOVE L8 (0 DUPLICATES REMOVED)
L11          20 S L3 (P) (L5 OR L6 OR L7)
L12           5 DUPLICATE REMOVE L11 (15 DUPLICATES REMOVED)
L13           5 S L12 NOT L10

=> log y
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FULL ESTIMATED COST                                   52.51      52.72

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
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CA SUBSCRIBER PRICE                                   -1.86      -1.86

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